Perspective

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Journal of Stroke Research & Therapy

2024
Vol.8 No.3:215

Innovative Nano Copolymer Applications and Advanced Approaches in Neurorehabilitation

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Received date: September 02, 2024, Manuscript No. IPSRT-24-19850; **Editor assigned date:** September 04, 2024, PreQC No. IPSRT-24-19850 (PQ); **Reviewed date:** September 17, 2024, QC No. IPSRT-24-19850; **Revised date:** September 24, 2024, Manuscript No. IPSRT-24-19850 (R); **Published date:** September 30, 2024, DOI: 10.36648/ipsrt.8.3.215

Citation: Jin X (2024) Innovative Nano Copolymer Applications and Advanced Approaches in Neurorehabilitation. Stroke Res Ther Vol.8. No.3:215.

Description

The goal of neurorehabilitation is to help patients with diseases of the central and peripheral nervous systems regain their function. Despite this, there are still few effective treatment options available. This work presents a novel nanocopolymer involving acrylamide and glycodeoxycholic acid that was created using reversible addition-fragmentation chain transfer polymerization. The reversible insoluble-to-soluble transition of this nanocopolymer in water occurs at a temperature that is associated with its Upper Critical Solution Temperature (UCST). This temperature is excellent for biological applications since it can be carefully regulated to about 37 °C. By creating a host-guest complex, the addition of β-Cyclodextrin (β-CD) modifies this transition temperature, increasing the flexibility of the copolymer. Loaded with compound 1, $CaCO_3$-PAAm-GDCA@1 reduced ferroptosis by modulating the Nrf2 and GPX4 pathways and markedly increased the proliferation of injured neuronal HT22 cells. These results demonstrate the potential of tailored nanocopolymers in improving neurorehabilitation therapy and provide a solid basis for the development of neuroprotective medications. Neuro-rehabilitation therapy should be started as soon as patients with Acquired Brain Injury (ABI) reach a minimal degree of clinical stability. Each required specialized treatment should result in 45-60 min sessions of rigorous therapy. It is imperative to have a well-organized, interdisciplinary staff with experience working with patients and their families.

Cardio metabolic disease

While outpatient treatment should begin when the patient's clinical condition permits and intensity criteria are satisfied, inpatient rehabilitation is advised for patients with severe impairments or those in the acute phase. The best available data should guide the customization of the treatment duration based on the patient's reaction and potential for future improvement. Support services, physical activity plans and follow-up care should be provided to patients after they are discharged in order to preserve benefits, identify problems and evaluate any functional changes that might call for further treatment plans. Adults with cerebral vascular disease are more likely to die and become permanently disabled, which puts a heavy burden on healthcare resources. Cerebrovascular disease development is significantly influenced by metabolic illnesses that arise from genetic predisposition and bad lifestyle choices. The field of cardiometabolic disease is one that is both well-known and fast developing. Numerous nations have adopted cardiometabolic medicine, which combines the treatment of cardiovascular and metabolic diseases. Comprehensive care of cerebrovascular illness and metabolic risk factors is still relatively new, nevertheless. The concept of cerebrometabolic disease is introduced, its possible pathogenesis is examined, recent integrative therapeutic approaches are summarized and future directions and challenges to improve the understanding and integrated management of metabolic and cerebrovascular issues are discussed in this minireview.

Mitochondrial homeostasis

Parkinson's Disease (PD) is mostly caused by mitochondrial malfunction, which also affects how the illness develops over time. Neuroprotective advantages could be obtained from interventions that attempt to preserve mitochondrial homeostasis and increase mitochondrial biogenesis. Apart from their fundamental role in the tricarboxylic acid cycle, mitochondria are involved in a number of other functions, including quality control, cellular metabolism and the generation of reactive oxygen species. Thus, it is essential to comprehend mitochondrial control and function in Parkinson's disease. In order to preserve mitochondrial equilibrium, this review seeks to promote research on neuroprotection techniques that target the mitochondria. First, we provide an overview of recent research on *AMBRA1, SYNJ2BP and SIAH3*, genes that control PD-related mitochondrial autophagy *via* PTEN-Induced Kinase 1 (PINK1). Next, we go over mitochondrial proteins linked to Parkinson's disease. The chronic illness known as Cubital Tunnel Syndrome (CTS) is brought on by compression of the ulnar nerve in the elbow. There are several places along the ulnar nerve's journey where it can become entrapped, the most frequent being the elbow joint. Muscle atrophy in the hand, weakness and numbness in the ulnar nerve's distribution area are typical symptoms. individuals with minor symptoms respond well to conservative treatment individuals with moderate to severe symptoms frequently need surgery. The complicated etiology of CTS and the peculiar structure of the ulnar nerve have resulted in less than ideal results, despite much study on the clinical therapy of the disorder. Physiotherapy is still essential for encouraging nerve regeneration and lowering rates of impairment.